EPA COMMENTS – AUGUST 25, 2016
REVISED DRAFT BASELINE HUMAN HEALTH RISK ASSESSMENT REPORT
FOR THE LOWER PASSAIC RIVER STUDY AREA
DATED DECEMBER 2015

No.	General Comment
1	The document will require revisions to address EPA comments that were not appropriately addressed from previous comments on the June 2014 draft BHHRA. EPA's comments must be incorporated appropriately; if they are not, the document will not be approvable and EPA will proceed as per Paragraph 44 of the Agreement. If the next draft of the BHHRA is deficient, EPA may elect to modify the document itself pursuant to Paragraph 44 of the Agreement, and, as per Paragraph 47 of the Agreement, the CPG would be required to accept the findings of the modified report (subject to dispute resolution).
2	Consistent with the Dispute Resolution (EPA letter 2/6/12, see page 3693 of the BHHRA Appendices pdf), all instances where it states "At the direction of USEPA Region 2" or "USEPA Region 2 directed the CPG to use" shall also include the phrase "and consistent with guidance and policies." Specific examples are provided below. • Page ES-5. "USEPA Region 2 has directed the CPG to use [footnote], and…" • Page ES-6. "At the direction of USEPA Region 2, the…" • Page 4-9. "… those that USEPA Region 2 directed the CPG to use …" • Page 4-10. "…those directed by USEPA for use…" • Page 7-7. "USEPA Region 2's directed exposure parameter…" • Page 7-10. "…fish consumption rates were directed by USEPA Region 2 (USEPA 2012b), and were…" • Pages 8-2 to 8-3. "…those that Region 2 directed the CPG to use…"
3	The text still uses the term "NCP threshold" which suggests a bright line for decisions at Superfund sites. The Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions, April 22, 1991 clearly states that the risk range is not a bright line. As indicated in previous comments, the presentation of risk should be presented as below the risk range, above the risk range or within the risk range, or above or below or equal to the goal of protection of a non-cancer HQ/HI of 1.
4	The Superfund Technical Support Center (STSC) letters of November 12 and 24, 2015 regarding surrogates for cis-nonachlor, oxychlordane, and trans-nonachlor were provided in EPA's December 4, 2015 letter, but were not incorporated in the revised draft because of time limitations in submitting the report. Specific comments provided below address adding the information to Section 5, Section 7, and Tables 5-1 and 5-2. Changes to these toxicity values will also impact risk calculation tables; noncancer hazards will increase slightly for the nonachlors and cancer risks will decrease for all three COPCs. Cis-nonachlor and oxychlordane will no longer be considered potential COCs for the LPRSA with the updated instructions for the relative potency factors.

No.	General Comment
5	For estimation of background risks associated with direct contact with sediment, the BHHRA only discussed cancer risks for comparison to site risks. For this exposure pathway, noncancer hazards were more of an issue for the site than cancer risks (i.e., cancer risks were less than 1×10^{-4} but HI was greater than 1), and background noncancer hazards should also be discussed in the text. (The noncancer hazards for background sediment were presented in a table in Appendix L, but not included in the evaluation in Section 6.5.2.)
6	The Uncertainty Evaluation section is very long (48 pages) and inclusive of potentially valid but secondary information. A meaningful uncertainty section is expected to be a balanced appraisal of major uncertainties that will significantly affect the site-specific numerical risks as they relate to the selection of remedies. There are uncertainty issues that do not need to be included and other uncertainties that should be reduced in size to a paragraph. Per EPA General Comment 12 on the Draft BHHRA (comments dated October 16, 2015), "the text requires revisions to concentrate on the main risk drivers with less emphasis on exposure parameters that are not significant drivers." The discussion of uncertainty needs to concentrate on risks above the NCP risk range and an HI = 1. Similarly, the Executive Summary should concentrate on the main risk drivers consistent with this recommendation. The Uncertainty Evaluation continues to discuss uncertainties in some assumptions without linking them to an impact on the site risk estimates. Examples: - Critique of default dermal absorption fractions for three sets of chemicals (pp. 7-26 to 7-29) when dermal contact with sediment was a very minor contributor to cumulative risks/hazards for the LPRSA. Indeed, for one of the chemical groups (i.e., PCBs), estimated cancer risks never exceeded 10^{-6} and noncancer hazards were well below an HI of 1. - Critique of default approach for estimating TCE cancer risks to non-adult receptors (p. 7-39) when TCE cancer risks never exceeded 10^{-6}. Detailed discussions of exposure parameters or chemicals that are not significant drivers distract the reader from issues that are key to interpreting the primary site risks and should be limited to a summary statement or removed from the report.
7	Summary sections of the report should include the magnitude of risk/hazard estimates (missing from ES.3 Conclusions and 8.2 Conclusions). Summary sections also should identify key target organs/effects potentially associated with the noncancer hazards (missing from ES.1 Summary of Key Findings, ES.3 Conclusions, and 8.2 Conclusions).

No.	General Comment
8	Multiple descriptions of correspondence and calls leading up to final assumptions applied in the risk assessment do not add value to the HHRA report and should be removed. Technical basis for values used should be provided in the main text and uncertainties in those values are discussed in the uncertainty section (Section 7). All correspondence between EPA and the CPG regarding the risk assessment between September 2010 and December 2015 are provided in Appendix M of the BHHRA. It is acceptable to provide the list of correspondence about exposure assumptions once (i.e., footnote 27 on page 4-10), but subsequent descriptions of communications should be removed: • Page 4-13, footnote 28 • Page 4-16, footnote 30 • Page 4-18, footnote 31 • Page 4-21, footnote 32 • Page 7-7, second complete paragraph
9	With regard to the Creel Angler Survey (CAS), the document details the attributes of the study, but fails to discuss potential issues with the representativeness of the CAS study. The document does, however, go into a substantial amount of detail questioning the default parameters and other surveys used as the basis of EPA's recommended exposure parameters (see section 7.2.1.2 for example). Discussion of the CAS study in the document should also include identification of potential issues of the CAS study. On page 7-12, last paragraph, the discussion about the fish consumption rates in the range of 1 meal/month to 2 meals/month are supported by the CPG's CAS. As per EPA's previous comments on the BHHRA including EPA's October 30, 2015 email from Stephanie Vaughn to Rob Law, this quantification is inconsistent with the direction provided by EPA and all references to the CAS should clearly state that the data represents current conditions, in the presence of a consumption advisory.

No.	Page No.	Specific Comment
10	Pages ES-1 and ES-2, Section ES.1 Summary of Key Findings	The text regarding the primary purpose of the risk assessment needs to be expanded to "inform the public regarding risks" in addition to the risk manager. The use of the term "threshold" is inconsistent with OSWER Directive 9355.0-30. Consistent with the Directive, a more appropriate term is "exceed the risk range". The discussion regarding the "dominant risk contributor" for the fish consumption pathway highlights TCDD toxicity equivalency and PCBs as the main risk drivers. The only other chemical with an HI > 1 is mercury. The discussion needs to clarify that the other contaminants e.g., pesticides, arsenic, BAP, are below the upper end of the risk range and below an HI = 1.
11	Page ES-2, Section ES.1 Summary of Key Findings	Bullets identifying noncancer health hazard estimates with a Hazard Index (HI) greater than 1 should also identify potential health effects (i.e., target organ effects) associated with that hazard estimate.

No.	Page No.	Specific Comment
12	Page ES-3, Section ES.1 Summary of Key Findings, Last bullet	The discussion of background should clarify that excluding TCDD-TEQ still results in a cancer risk greater than the risk range and a non-cancer HI > 1.
13	Page ES-4, Section ES.2.1 Data Evaluation and Hazard Identification	**Paragraph 1.** Recommend removing statement "as agreed with USEPA Region 2" and "CPG's RI/FS" programs requires consideration since this language suggests that this is not an EPA document. **Paragraph 2.** Change sentence "Because of the conservative screening process that was used …" to "The screening process used to identify COPCs is designed to assure that chemicals not identified as COPCs are minor contributors to the overall risks and hazards from the site."' **Paragraph 3**. Remove the first sentence beginning "Many of the chemicals identified as COPCs…" as it is broad and conclusory.
14	Page ES-5, Section ES.2.2 Exposure Assessment	Remove term "conservative." The more appropriate term is "health protective" and should be used throughout the document.
15	Page ES-6, Section ES.2.2, Exposure Assessment	First full paragraph: Insert the following at the end of the first sentence "because even if the consumer does not eat the hepatopancreas, exposure to the chemical may still occur if the crab is cooked before the hepatopancreas is removed."
16	Page ES-7, Table ES-1	Add USEPA 2014 to footnote d.
17	Page ES-11	*Fish Consumption*. The discussion of the cancer risks should clarify whether the other risk contributors e.g., about 4% were above the risk range or not. *Fish Consumption and Crab Consumption* Here, and throughout the document, remove the term "target endpoint" and use the term "target organ effect" consistent with terminology used in RAGS Part A (EPA 1989). *Direct Contact with Sediment and Surface Water* Remove the term "thresholds" and replace with "range or noncancer HI = 1".

No.	Page No.	Specific Comment
18	Page ES-12, Section ES.2.5 Identification of Potential Chemicals of Concern	Remove the term "thresholds" and replace with "range or noncancer HI = 1". Replace the text before the table with: "The following table summarizes potential COCs with individual pathway cancer risks greater than 10^{-4}, and/or an individual pathway noncancer hazard quotient (HQ) greater than 1." Remove chemicals with a cancer risk $<10^{-4}$ and noncancer HI<1 from the summary table and revise the footnotes accordingly (i.e., delete footnotes c and d). Please revise this section to indicate that details regarding other chemicals within the risk range and below a HI=1 are provided in Section 6.4. Replace the text after the table with: "These potential COCs are also present in upstream and regional background media. The levels of these COCs in background fish and/or crab tissue were found to pose consumption risks/hazards above the NCP risk range or noncancer HI=1. For methyl mercury, the background concentrations in fish tissue and the corresponding hazards are comparable to or greater than in fish collected in the LPRSA."
19	Pages ES-12 to ES-15, Section ES.3 Conclusions	Replace Section ES.3 with the revised text provided in Attachment A. Note that comments to this section of the Executive Summary also apply to Section 8.2 Conclusions (pages 8-8 through 8-10), which has text that almost exactly matches, and should also be replaced. Issues with the Conclusions section: The conclusions of the Executive Summary should specifically identify the calculated risk and HI values and not just note that values are above NCP risk/hazard thresholds (e.g., first bullet) or some degree lower than an alternate approach (e.g., fifth bullet). In addition, the text concentrates on the percentage contributions of the chemicals, but should also clarify which chemicals are above the risk range or HQ = 1. EPA notes that the last bullet on pages ES-14 and 8-10, does identify risk and hazard values for background levels. Text in the conclusions summarizing the site risks should be equally transparent. The section should briefly identify potential health effects (i.e., target organ effects) associated with the noncancer hazards exceeding an HI = 1. Since the PCB toxicity approach has a minimal impact on cumulative risks/hazards, the summary of this topic in the conclusions should be removed. The final paragraph of the section includes a phrase that does not make sense as written (i.e., "pose risks that contribute significantly to LPRSA risks"). Risks estimated for receptors in one area do not contribute to risks to receptors in another area. The sentence in Attachment A has been revised to "Upstream and regional levels of several potential COCs, including PCBs, pesticides, PAHs, and mercury, are elevated and may contribute to levels observed in the LPRSA and to risks estimated for LPRSA receptors."

No.	Page No.	Specific Comment
20	Page 1-1, Section 1.0 Introduction, Second Paragraph	The second sentence of this paragraph, starting with "Using the data…," should be removed. Change last sentence to: USEPA (2014a) provides standard default exposure assumptions (e.g., parameters for age-specific body weight, skin surface area, dermal absorption, etc.) that can be used at sites based on the Exposure Factors Handbook (2011) in the absence of site-specific information.
21	Page 2-2, Section 2.1.1 Site Background, Second Complete Paragraph	In addressing EPA Specific Comment 32 on the Draft HHRA (10/16/15), text was added about the removal action at RM 10.9. In the revised text, the final sentence of the paragraph states that sediments at RM10.9 were removed "to address high concentrations of dioxins and other contaminants found at the surface of sediments in this area." This implies that the high concentrations were just at the surface and have been addressed. However, as part of the removal action, the area has a cap overlying the remaining contaminated sediment. For completeness, please add the following statement to the end of this paragraph: "In addition, as part of the removal action a cap was placed over remaining contaminated sediments in this area."
22	Page 2-2, Section 2.1.1 Site Background, Last Paragraph in Section	Per response to EPA Specific Comment 33c (10/16/15) on the Draft HHRA, add a reference to the RI report in the final sentence about regional conditions.
23	Page 2-6, Section 2.3 River Use	As discussed in EPA Comment 39 (10/16/15) on the Draft HHRA, the discussion of fishing should also recognize the potential for exposures under future conditions. Reference to NJDHSS requires update to the New Jersey Department of Health (now NJDOH).
24	Page 2-7, Footnote 10	Add the following to the end of footnote 10: ", but did include five results from Newark."
25	Page 2-8, Footnote 11	Change the wording to: USEPA Region 2 did not provide input …
26	Page 2-9, Section 2.3.1.1	Last sentence: Add the following after the last sentence of this section "Results of this study have not been published in the peer-reviewed literature."
27	Page 3-1, Section 3.1 Data Evaluation	The discussion in the last paragraph regarding the Cal EPA Air Resources sampling method needs clarification. Need to clarify whether the data was QA/QCed and if Edison had any concerns about this method.

No.	Page No.	Specific Comment
28	Page 4-4, Section 4.1 Human Health Conceptual Site Model	In the third full paragraph on page 4-4, regarding the inhalation pathway, change "30 years" to "26 years."
29	Page 4-9, Section 4.3	Replace the beginning of last sentence of second paragraph with the following: "While risk management decisions are based on the RME, the purpose of evaluating both an RME and a CTE…"
30	Page 4-13, Section 4.3.6.1	Remove footnote 28. Add ", included in Appendix M of this BHHRA" to the reference at the end of the first sentence of Section 4.3.6.1.
31	Page 4-14, Section 4.3.6.1 Fish Ingestion Rate, Second Bullet	Define Newark Bay Complex either in the bullet or in a footnote on this page, "The Newark Bay Complex study area from Burger (2002) included Newark Bay and tidal portions of the Hackensack River, Arthur Kill, and Kill van Kull."
32	Page 4-16, Section 4.3.6.3, Cooking Loss for Fish	Remove footnote 30. The technical information is provided in the text of Section 4.3.6.3, and all correspondence is provided in Appendix M of the BHHRA.
33	Page 4-17, Section 4.3.6.5, Cooking Loss for Crab	The 2013 document citing NJDHSS, should indicate that NJDHSS is now NJDOH.
34	Page 4-18, Section 4.3.6.5, Cooking Loss for Crab	Remove footnote 31. The technical information is provided in the text of Section 4.3.6.5, and all correspondence is provided in Appendix M of the BHHRA.
35	Pages 4-19 and 4-20, Section 4.3.7.3 Body Surface Areas in Contact with Sediment and Surface Water	Skin surface areas for adults were based on means rather than 50th percentiles as accurately identified in the tables; the description in the text should be corrected. Replace "50th percentile" with "mean values" in the third, fifth, and sixth paragraphs of this section.
36	Page 4-21, Section 4.3.7.4, Sediment to Skin Adherence Factors	Remove footnote 32. The technical information is provided in the text of Section 4.3.7.4, and all correspondence is provided in Appendix M of the BHHRA.

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37	Page 4-26, Section 4.3.9 Body Weight	The revised body weight for young children was not based on a standard default, but derived from values in the 2011 Exposure Factors Handbook as shown in Appendix N. The description in the text should be corrected as follows: a. Second sentence, remove phrase "and 17 kg for young children" b. Third sentence, change "Body weights for adolescent age groups…" to "Body weights for young children and adolescent age groups…" Fourth sentence, add "17 kg for the 1 to <7 year old young child," to the list.
38	Page 4-27, Section 4.3.10.2, Oral Absorption Adjustment Factors	Second paragraph: Change "The assumption of 100% RBA results in an overestimate of risk…" to "The assumption of 100% RBA would result in an overestimate of risk…"
39	Pages 4-31 to 4-32, Section 4.4.4.1 EPCs for 2,3,7,8-TCDD in Surface Water	Add the following footnote to the end of the second sentence: A split sample of 11A-CE04-TTR1 was also collected and analyzed separately, and did not confirm the elevated concentration. The split sample result was 81 times lower.
40	Pages 5-2 to 5-3, Section 5.1	The fifth paragraph of this section (last paragraph on page 5-2 and top of page 5-3) should be removed because it does not reflect the current IRIS process that was noted in the second paragraph. IRIS is not updated on a monthly basis and the Verification Workgroup was disbanded 20 years ago.
41	Page 5-3, Section 5.1 Sources of Toxicity Data	The discussion of HEAST is not necessary. HEAST is clearly identified as a Tier 3 Toxicity value so it is not necessary to restate the reasoning for identifying this chemical as a Tier 3. The discussion of the toxicity value for Thallium needs to clarify that value is based on Thallium Soluble Salts. Also, this is an Appendix value indicating limitations on its use. The text on page 5-6 regarding these values should be referenced for this chemical.

No.	Page No.	Specific Comment
42	Page 5-4, Section 5.1 Sources of Toxicity Data, First Full Paragraph and Table	Per response to EPA Specific Comment 87c (10/16/15) on the Draft HHRA and included in December 4, 2015, letter to CPG, two additional STSC references for surrogate values should be included in the last sentence of the paragraph. These should also be added to Section 9.0 References. a. USEPA 2015: Letter from Superfund Technical Support Center to Marian Olsen dated November 12, 2015. Clarification on the use of male or female relative potency factors to derive surrogate points of departure. b. USEPA 2015: Letter from Superfund Technical Support Center to Marian Olsen dated November 24, 2015. Inquiry as to whether the cancer risks of chlordane should be evaluated and if relative potency factors can be applied on the finding of hypertrophy for nonachlor. In addition, based on these letters, chlordane relative potency factors should apply only to the noncancer assessment. In the table on page 5-4, change "Chlordane (IRIS) with RPF" to "Chlordane (IRIS)" in the CSF column for cis-Nonachlor, Oxychlordane, and trans-Nonachlor. As noted in the CPG's December 10, 2015 email, updating these toxicity values has minimal impact on final noncancer hazard estimates, but more significant impact on cancer risk estimates. Cis-nonachlor and oxychlordane will no longer be considered potential COCs for the LPSRA with the updated instructions for the relative potency factors.
43	Page 5-4, Section 5.1 Sources of Toxicity Data, Paragraph after Table	The statements regarding the quality of toxicity values is inaccurate and should be removed. The hierarchy provides adequate information regarding toxicity values and further discussion is not needed. Specifically, ATSDR values are externally peer-reviewed and EPA coordinates with ATSDR. This text should be dropped.
44	Page 5-4, Section 5.2 Noncarcinogenic Toxicity Assessment	Replace the term "true threshold" with "threshold."
45	Page 5-5, Section 5.2 Noncarcinogenic Toxicity Assessment, Second paragraph	Not clear why the term "In regulatory toxicity assessment" is used. Remove this phrase.
46	Page 5-6, Section 5.2 Noncarcinogenic Toxicity Assessment	The text regarding C9-C18 requires clarification that this value is a surrogate value for initial evaluation and needs to be updated with information provided by NCEA.
47	Page 5-6, Section 5.3 Carcinogenic Toxicity Assessment	The text regarding the classifications of carcinogens based on the 1986 Cancer Guidelines needs to clarify that these classifications are being used until the chemicals are reassessed under the IRIS program based on the 2005 Cancer Guidelines.

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48	Page 5-7, Section 5.3 Carcinogenic Toxicity Assessment	With regard to "narrative descriptions" in the second full paragraph on this page, replace the phrase "has not generally been implemented for chemicals" with "has not yet been implemented for many chemicals." As discussed above, inclusion of narratives requires a re-evaluation of the chemical as part of the IRIS program.
49	Page 5-8, Section 5.3 Carcinogenic Toxicity Assessment	Paragraph 2: Third sentence, remove the phrase "as that is the value used in the RSL tables (USEPA 2015b)." Third sentence should read "… a value meeting Tier 3 criteria developed by NJDEP…" Fifth sentence, remove the phrase "As noted in the user's guide for the RSLs (USEPA, 2015b),"
50	Page 5-11, Section 5.5.1, Dioxins and Furans	Include reference to U.S. EPA 1996 regarding the CSF for dioxin of 150,000.
51	Page 5-15	Remove mention of RSLs as a source of toxicity values. The hierarchy should be used. Reference EPA's 1993 Relative Potency Evaluation for PAHS as the source of the carcinogenic PAH toxicity values.
52	Page 6-1, Footnote 40	Add the following to the end of the footnote "However, ORD/NCEA is re-considering the appropriateness of updating this factor for purposes of calculating lifetime average daily dose, and the standard default exposure assumption for lifetime remains 70 years (USEPA 2014a)."
53	Page 6-1, Section 6.1 Carcinogenic Risk Characterization	Remove the discussion regarding background cancer risk levels based on the American Cancer Society.
54	Page 6-3, Section 6.2 Noncarcinogenic Risk Characterization	Change from "noncarcinogenic risks" to "noncarcinogenic hazards." Change title to Noncarcinogenic Hazard Characterization. Please remove "NCP" before goal of protection in the last paragraph of Section 6.2. The NCP specifically addresses the risk range and not the noncancer hazard. Please also make this same change to the second bullet on page 6-25 and anywhere else in the document this phrase has been used.

No.	Page No.	Specific Comment
55	Page 6-3 and 6-4, Section 6.2.1 Risk Characterization for Lead	Page 6-3, Second sentence of Section 6.2.1: Change "target blood lead level" to "USEPA's blood lead level of concern." Page 6-3 to 6-4, Third sentence of Section 6.2.1: change "USEPA regulatory target" to "USEPA risk reduction goal." Footnote 41: Change "Centers for Disease Control (CDC)" to "Centers for Disease Control and Prevention (CDC)"
56	Page 6-4, Section 6.3 Risk Characterization Results	Please revise the first sentence of the first paragraph to read as follows: The results of the risk characterization are presented below by receptor, highlighting risks exceeding 10^{-4} and/or a non-cancer HI greater than 1.
57	Page 6-4, Section 6.3.1 Recreational Angler	The discussion of crab consumption needs to acknowledge that Burger did identify crab consumption in the survey that was used to derive the consumption rate. Add the following to the end of the first paragraph: "Crab consumption rates assumed in this evaluation are based on anglers who catch and consume crabs from the Newark Bay Complex, which includes tidal portions of rivers (Burger 2002)."
58	Page 6-5, Section 6.3.1.1 Recreational Angler – Young Child	Remove "applicable NCP benchmarks". Please replace the "NCP risk range and the goal of protection of an HI=1"
59	Page 6-24, Section 6.3.6 Lead Risk Characterization	The adult lead methodology available at: https://www.epa.gov/superfund/lead-superfund-sites-frequent-questions-risk-assessors-adult-lead-methodology should be cited in place of the reference to Bowers et al. (1994). The Adult Lead Methodology documents are the basis for the evaluation of lead exposures to adults.
60	Page 6-24, Section 6.3.7 Risk Characterization Summary	The Risk Characterization Summary should specifically identify the risks exceeding the risk range and goal of protection for non-cancer and the associated chemicals. The reader should not be referred to a Table to find the results of the assessment. At a minimum the key risk pathways should be identified before the discussion of the relative percent contributions of the individual chemicals to the total risk or hazard.
61	Page 6-30, Section 6.4 Potential COC Identification	Remove term "target endpoint" and replace with "target organ effect."

No.	Page No.	Specific Comment
62	Page 6-31, Potential COC Identification, Unnumbered Table	The third paragraph on page 6-30 indicates that for each medium and exposure route, potentially carcinogenic potential COCs are presented in these potential COC summary tables according to the following cancer risk range categories: greater than 10-4, greater than 10-5 and less than 10-4, greater than 10-6 and less than 10-5. And an HI greater than 1 and an HI greater than 0.1 and less than 1. However, the unnumbered table on page 6-31 is not a clear presentation of the potential COCs and the media of concern that exceed the risk range and the noncancer goal of protection of an HI=1. In order to address this, please replace the summary table on page 6-32 with a table that includes chemicals by media greater than 10^{-4}, greater than 10^{-5} and less than or equal to 10^{-4}, greater than 10^{-6} and less than or equal to 10^{-5}. And an HI greater than 1 and an HI greater than 0.1 and less than 1 in this section.
63	Page 6-32, Section 6.4 Potential COC Identification	The last paragraph of Section 6.4 on page 6-32 includes information not necessary for the risk characterization section of the BHHRA. EPA has provided language to replace this paragraph below: Please revise the last paragraph of Section 6.4 to read as follows: Additional factors considered in the identification of potential COCs include contributions from background sources described below. Section 6.5 provides details regarding this evaluation. In addition, overall uncertainties associated with the four steps of the risk assessment process that may also be considered in the evaluation of potential COCs are provided in Chapter 7 of the BHHRA.
64	Page 6-33, Section 6.5 Background Evaluation	Remove term "target endpoint" and replace with "target organ effect."
65	Page 6-34, Section 6.5.1 Summary of Regional Background Data Sets, Table	Correct the number of accessible surface sediment samples from the 2008 LRC Program from "6 samples" to "2 samples", consistent with the number of data points from this program used in Appendix L.
66	Page 6-34, Section 6.5.2 Regional Background Risk Evaluation, Third Bullet and Footnote 43	For estimation of background risks associated with direct contact with sediment, the BHHRA only discusses cancer risks for comparison to the LPRSA. For this exposure pathway, noncancer hazards were more of an issue for the LPRSA than cancer risks (i.e., cancer risks were less than 1×10^{-4} but HI was greater than 1), and should also be included in the comparison to background. Change the end of the third bullet from "cancer[43]" to "cancer and noncancer" and remove footnote 43.

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67	Pages 6-39 to 6-40, Section 6.5.2.4 Regional Background Risks for Direct Contact with Surface Sediment	Add a noncancer assessment to this section. a. Remove the last sentence of the paragraph just before the sediment risk table on page 6-39. b. Add a subsection for noncancer sediment hazards on page 6-40.
68	Page 6-40, Section 6.5.2.5 Summary of Regional Background Risks	Remove the phrase "risks posed by" from the first sentence. It should state that "the levels of potential COCs … pose cancer risks…" not that "… the risks posed by the levels of potential COCs … pose cancer risks…" Change the discussion in the second paragraph to include consideration of noncancer hazards from direct contact with sediment rather than just cancer risks.
69	Page 7-1	Replace "due to lack of absolute scientific knowledge" with "due to both variability and uncertainty in exposure patterns of human receptors and toxicity of chemicals." Remove the term "regulatory" from "regulatory risk assessment."
70	Page 7-7, Exposure Scenario Assumptions	Second complete paragraph: Consistent with General Comment 8, remove the first two sentences of this paragraph, from "USEPA Region 2's directive…" through "…(USEPA 2014a)."
71	Page 7-7, Section 7.2.1.1 Sediment and Surface Water Exposures	Add the following sentences after the first paragraph: "As noted in Section 6, direct contact with sediment and surface water are minor contributors to total cancer risks, posing sitewide and segment-specific risks within or below the NCP risk range. Similarly, direct contact with these media are minor contributors to cumulative noncancer hazard, posing sitewide and segment-specific HIs below 1, with the exception of RM 6-9 and RM 6-9 East Bank in particular."
72	Page 7-9, Section 7.2.1.1 Sediment and Surface Water Exposures	Remove "NCP benchmarks". Use term "NCP risk range" and for non-cancer refer to exceeding the goal of protection of a HI=1.
73	Page 7-9, Section 7.2.1.2 Fish and Crab Consumption Exposures, First Paragraph	Per response to EPA Specific Comment 124 (10/16/15) on the Draft HHRA, add text here stating that urban populations often have less opportunity to travel to more desirable locations for recreation.

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74	Pages 7-10 through 7-13, Section 7.2.1.2 fish and Crab Consumption Exposures	Per response to EPA Specific Comment 127a (10/16/15) on the Draft HHRA, the text needs additional clarification that the Burger survey was for the Newark Bay Complex and not Newark Bay alone. These pages still mention "Newark Bay trips," "Newark Bay fish consumption," and "Newark Bay anglers." Locations surveyed by Burger (i.e., the Newark Bay Complex) also included tidal portions of waterways adjacent to Newark Bay.
75	Page 7-10, Fish Consumption Rate, Second Paragraph, Fourth Sentence	Change "A total of 61 consuming anglers in the Newark Bay Complex were intercepted once…" to "A total of 65 consuming anglers in the Newark Bay Complex were intercepted and interviewed once…" A total of 65 anglers were interviewed and the number dropped to 61 anglers only after USEPA removed 4 outliers. Add a footnote after the edited phrase: "Burger (2002) noted that they saw the same people at the survey locations from time to time but each person was interviewed only once for the study."
76	Page 7-10, Fish Consumption Rate, First Bullet	The mean portion size noted here of 11.7 ounces was reported in Burger (2002), but does not reflect the mean portion size in the data used to estimate the fish consumption rate after outliers were removed. a. The second sentence should be revised to "… mean portion size reported by consumers *in Burger (2002)* of 11.7 ounces…" (text italicized here to indicate addition). b. In addition, add the following text after the second sentence: "USEPA's analysis of the raw Burger (2002) data identified and excluded four records because the respondents estimated a serving size greater than 30 ounces per meal. The mean portion size was 7.45 ounces for the 61 respondents from the Burger (2002) raw data that were used to estimate the fish consumption rates in this report; this portion size is consistent with the other surveys mentioned above."
77	Page 7-12, Table	Per response to EPA Specific Comment 127b (10/16/15) on the Draft BHHRA, a table of fish ingestion rates used in other Region 2 HHRAs has been added to the report. However, this table is limited to just four recent sites and presents an incomplete picture. Figure 3 from the Fish and Crab Consumption Rates memo (USEPA 2012; page 3709 in the Appendices pdf file) has a more complete listing, showing values for 15 sites in Region 2 going back to 1990. Refer the reader to the figure for additional information. In addition, add a footnote below the table: Ingestion rates of 25 and 26 g/day in the table were based on a recommended default fish ingestion rate from USEPA 1997 that is no longer recommended as a default in USEPA 2011.
78	Page 7-12, Fish Consumption Rate, Last Paragraph, Fourth Sentence	This sentence references the BHHRA for the Lower Duwamish River. However, the consumption rate assumed for a site in Washington State (Lower Duwamish River) is not directly relevant to a site in the northeast region of the United States. As stated in the Exposure Factors Handbook (USEPA 2011) with regard to fish intake, "…available data are limited to certain geographic areas and cannot be readily generalized to the U.S. population of freshwater recreational anglers as a whole… For example, factors associated with water body, climate, fishing regulations, availability of alternate fishable water bodies, and water body productivity may affect recreational fish intake rates." Remove the sentence (fourth sentence of paragraph).

No.	Page No.	Specific Comment
79	Page 7-13, Crab Consumption Rate	First paragraph, last sentence, replace with "There is uncertainty in this ingestion rate." In addition, as previously noted, references to the area of the Burger (2002) study should state "Newark Bay Complex" and not just "Newark Bay."
80	Page 7-14, Crab Tissue Type Consumed	This subsection just mentions how risks are expected to change with assumptions about crab tissue type consumed, but these risks are actually quantified later in the report. Move the second paragraph from page 7-17 (starts with "Many anglers consume only the crab muscle…") to this section. Also, identify the HI values that exceed 1 in the moved text
81	Page 7-15, Section 7.2.1.2 Fish and Crab Consumption Exposures, Cooking Loss	Paragraph following table. In the sixth sentence, beginning with "Despite the variability…" change "…cooking loss factor in the assessment…" to "…cooking loss factor in the CTE assessment…"
82	Page 7-18, Section 7.2.1.4, Consumption of Other Biota	In Paragraph 1, please add the following sentence after the first sentence on this page. "Some of these biota, such as ducks and turtles, are fattier than fish or crabs and therefore may carry heavier burdens of PCBs/TCDD."
83	Page 7-22, Section 7.2.2.2 Uncertainty in Sediment EPCs	The discussion of the sediment EPCs based on a one mile segment requires further clarification. Please note "in Three-Mile Segment" in the final column of the table. Add the following to the text just before the final sentence of this section: "Similar results for one-mile segments are expected for the other receptors with sediment direct contact exposure (e.g., adolescent waders and swimmers, young child waders)."
84	Pages 7-25 to 7-30, Section 7.2.3 Estimation of Exposure Dose	The text should also indicate EPA's process and guidance that allows the evaluation of relative bioavailability of chemicals; however, data on bioavailability for the COPCs was not available to allow the modifications in bioavailability as was done for arsenic. In the first sentence, insert "where data are available," before "…absorption adjustment factors…" This whole section, including subsections 7.2.3.1 and 7.2.3.2, focuses on issues of uncertainty in bioavailability from sediments (both dermal and oral), without putting those issues in the context of site risk estimates: direct human contact with sediment, whether through dermal contact or incidental ingestion, is a relatively minor contributor to total risk for the LPRSA . For sediment exposures, cancer risks did not exceed the NCP risk range and noncancer hazard estimates only exceeded the goal of protection of an HI of 1 in a limited section of the river (i.e., RM 6-9, with maximum HI of 5), primarily due to TCDD-TEQ. The introduction to this section should provide this context. EPA would accept editing this section as indicated in these comments or removing it completely because it does not have bearing on the most significant risks for the LPRSA.

No.	Page No.	Specific Comment
85	Pages 7-26 to 7-29, Section 7.2.3.1 Default Dermal Absorption Fractions	This section should discuss this topic in the context of the risks/hazards for the LPRSA. Make the following edits to this section: a. Change the second sentence in this section (page 7-26) to "The default DAF for PAHs may be overestimated and a lower DAF could be used for TCDD-TEQ for areas with high f_{oc}." b. Insert the following after the second sentence: "Using the default DAFs, no dermal exposures to LPRSA sediment contributed significantly to estimated cancer risks or noncancer hazards. Cancer risks from sediment exposures were all below 10^{-4}, and primarily from incidental ingestion. Noncancer hazards from sediment exposure only exceeded an HI of 1 in RM 6-9 and RM 6-9 East (HI of up to 5), again primarily from incidental ingestion. Even in these areas, dermal HIs were less than or equal to 1. Estimated cancer risks and noncancer hazards from dermal exposure to sediment could be even lower in non-default DAFs are considered. c. Remove the phrase "and oral absorption" from the next sentence (previously the third sentence) because this section focuses on dermal absorption factors. d. TCDD-TEQ – Add the following text after the table on page 7-27: "While a lower DAF may be applicable if accessible areas with sediment f_{oc} > 10% are found, it is important to note that estimated cancer risks and noncancer hazards from dermal exposures to TCDD-TEQ in sediment are already within the NCP risk range and less than or equal to the goal of protection of an HI of 1." e. PCBs – Remove this subsection from pages 7-27 to 7-28. Cancer risks from dermal contact with PCBs in sediment never exceeded 10^{-6} and noncancer hazards were well below an HI of 1. f. PAHs – Add the following text at the end of this subsection on page 7-28: "However, it is important to note that estimated cancer risks and noncancer hazards from dermal exposures to PAHs in sediment are already within the NCP risk range and below the goal of protection of an HI of 1."
86	Page 7-29, Section 7.2.3.2 Oral Bioavailability	As noted in Comment 146 (10/16/15) on the Draft BHHRA, EPA continues to be concerned with presentation of scientific studies that have not been reviewed by the agency to support oral bioavailability factors for chemicals other than arsenic, especially for chemicals that are not even identified in the BHHRA as potential COCs for direct contact with sediment (i.e., PCBs and arsenic). See Attachment B for revised text for this section that is to be incorporated in the revised draft BHHRA.

No.	Page No.	Specific Comment
87	Pages 7-30 through 7-39, Section 7.3 Toxicity Assessment through Section 7.3.3	EPA's previous comments highlighted that the text should reference the Cancer Guidelines and the non-cancer RfD/RfC Guidance. Changes are recommended based on the Cancer Guidelines and RfD/RfC guidance and the updates to the IRIS agenda regarding the reassessment of cancer toxicity of dioxin. The issue is that although the revised text quoted the documents it also included information that is contradictory to what is said in the EPA Guidance/Guidelines. At this point, as the document is going final, the text should be consistent with EPA's Guidance/Guidelines. See Attachment C for revised text for this section that is to be incorporated in the revised draft BHHRA. Additional comments regarding some of the revisions to text within this section are provided below.

No.	Page No.	Specific Comment
88	Page 7-30, Section 7.3 Toxicity Assessment, Paragraph 1	In Paragraph 1, the statement regarding the Cancer Guidelines indicates the evaluation of the cancer slope factor only. The text needs to also indicate the evaluation of the Weight of Evidence for Carcinogenicity as part of the process and as noted in the Cancer Guidelines. The text indicates "and effects assumed to be without a threshold (potentially carcinogenic), although there is increasing scientific evidence that many carcinogens also act via a threshold mechanism." The term "threshold" is inaccurate. Footnote #3 of the Cancer Guidelines indicates the term "linear" is used consistent with the Guidelines in place of the term "threshold". The Guidelines text also indicates that "Estimating thresholds can be problematic; for example, a response that is not statistically significant can be consistent with a small risk that falls below an experiment's power of detection." The Cancer Guidelines do not support the conclusions presented in the LPRSA revised draft BHHRA (December 2015) that "there is scientific evidence that many carcinogens also act via a threshold mechanism." Further the guidelines indicate: "The Agency's more current guidelines for these effects (U.S. EPA 1996a, 1998b), however, do not use this assumption, citing the difficulty of empirically distinguishing a true threshold from a dose-response curve that is nonlinear at low doses." It is recommended that the text indicate: "The Cancer Guidelines highlight the "difficulty of empirically distinguishing a true threshold from a dose-response that is non-linear at low doses". Alternatively, this text can be dropped since we do not have non-linear toxicity values in the LPRSA assessment – the only mention is chloroform, later in this section, which is not a potential COC. The sentence regarding overestimates of risks is inconsistent with the Cancer Guidelines. Specifically, the Guidelines state: "The use of upper bounds generally is considered to be a health-protective approach for covering the risk to susceptible individuals, although the calculation of upper bounds is not based on susceptibility data. Similarly, exposure during some lifestages can contribute more or less to the total lifetime risk than do similar exposures at other times. The dose-response assessment characterizes, to the extent possible, the extent of these variations." Revisions to Paragraph 1 of Section 7.3 based on the Cancer Guidelines are provided in Attachment C.

No.	Page No.	Specific Comment
89	Pages 7-30 to 7-31, Section 7.3 Toxicity Assessment, Paragraphs 2 through 4	These paragraphs were in Section 5.1 in the June 2014 Draft BHHRA and moved to the uncertainty section, per response to Comment 84 (10/16/15). However some edits and additional information should be provided based on the relevant guidance mentioned in that comment/response. **Paragraph 2**: This paragraph focuses on limitations in the application of animal study results to predicting human dose-response relationships. The Cancer Guidelines (USEPA 2005b) provide additional insights into how animal study information is weighed by EPA, and some points from the guidelines should be added here. **Paragraph 3**: Change from term conservative to "health protective." Remove reference to "Sections 5.3 and 5.4 below" which is artifact from the text's earlier location in the report. **Paragraph 4**: The text refers to a 1989 guidance and needs to be updated to reflect the current guidelines/guidance. See Attachment C for revised text for this section that is to be incorporated in the revised draft BHHRA.
90	Pages 7-34 through 7-39, Section 7.3.3 Uncertainty in TEF Approach	Nearly 5 pages of the uncertainty section are devoted to discussing the TCDD TEQ Approach (USEPA 2010) as applied to dioxin and PCB data for this project. It is agreed that areas of uncertainty exist within the TCDD TEQ Approach. However, missing from the text is the acknowledgement that this approach, since first introduced in the 1980's: • Has been the focus of intensive scientific scrutiny • Has been improved and strengthened over the years by incorporating newer scientific studies as they became available and through World Health Organization (WHO) consensus regarding congener-TEF assignments provided by leading experts regarding toxicity of dioxin and dioxin-like compounds (DLCs) • In current form, is considered standard practice nationally and internationally for use in risk assessments involving dioxin and DLCs In short, the TCDD TEQ Approach has substantial scientific standing and is considered the best tool available for assessment of dioxins and DLCs in CERCLA risk assessments. Section 7.3.3 must affirmatively acknowledge the validity and applicability of the TCDD TEQ Approach for use in the subject BHHRA. See Attachment C for revised text for this section that is to be incorporated in the revised draft BHHRA.

No.	Page No.	Specific Comment
91	Page 7-39 to 7-40, Section 7.3.4 Potential Contribution from Early-life Exposures to Lifetime Risk	Paragraph 1, First Sentence: Remove phrase "and infant (0-1 yr)" from the first sentence because the ADAF approach includes an infant of 0 to <2 years. Paragraph 1, after first sentence: The rest of the first paragraph criticizes the default approach for estimating TCE toxicity to non-adult receptors, without putting this uncertainty into context for this LPRSA. Estimated cancer risks from exposure to TCE at the LPRSA never exceeded 10^{-6}. Remove all of this paragraph after the second sentence. Paragraph 2, Final Sentence: The following text requires revisions. "While there is uncertainty in the extent of early life exposures, the available data suggest that in utero and infant exposures to bioaccumulative COPCs via the mother's consumption of LPRSA fish and crab are not contributing appreciably to lifetime risk." Replace the sentence with the following: "The extent to which women of childbearing age are consuming or will consume LPRSA fish and crabs is uncertain."
92	Page 7-40, Section 7.3.5 Use of Surrogate Values	The following sentence needs revision: "The COPCs that required surrogates generally consist of chemicals/groups where the assignment of surrogates is generally accepted, including PAH compounds, DDx isomers, chlordane isomers, endosulfan isomers, butyltins, and TPH ranges." Within this sentence, replace the phrase "where the assignment of surrogates is generally accepted" with the phrase "that have been reviewed by the STSC, and for which the STSC has developed specific surrogate recommendations."
93	Page 7-40, Section 7.3.6 Tier 3 Toxicity Values	Replace the text in this section prior to the table with the following: "There is uncertainty associated with the toxicity values based on Tier 3 sources due to the variable nature of peer-review and consensus among scientists on the best estimate of toxicity. While most COPCs have Tier 1 or 2 toxicity values, it was necessary to identify Tier 3 toxicity values for six COPCs: organic arsenic, copper, thallium, TPH C9-C18, hexavalent chromium, and TCDD-TEQ. The following table summarizes the relevant exposure and toxicity information for these six compounds; their contribution to the risk results is discussed below."
94	Page 7-43, Section 7.4 Risk Characterization	In Paragraph 1, change "…upper-bound exposure estimates…" to "…upper-bound and average exposure estimates…"
95	Page 7-43, Section 7.4.1 Risk from Multiple Chemicals	In Paragraph 2, cancer slope factors are mischaracterized as "upper 95th percentile estimates on a COPC's carcinogenic potency" and "upper 95th percentiles of probability distributions." Correct the description to "upper bound estimates of a COPC carcinogenic potency" throughout Paragraph 2.

No.	Page No.	Specific Comment
96	Page 7-44, Section 7.4.1, table	Remove arsenic from this table as an example since it is associated with other types of tumors including liver, etc.

No.	Page No.	Specific Comment
97	Pages 7-44 to 7-45, Section 7.4.2	Change the language in the first sentence to: Generally, the goal of a risk assessment is to estimate risk to the RME individual. Third sentence: Change "extremely conservative (health-protective)" to "health protective, and the majority of people will have a lower level of potential risk." Remove the rest of the paragraph, starting with "For example, …" The example would only be accurate if all the input variables have the same variability and shape, which is rarely the case in actual situations as discussed in EPA's 2004 Office of the Science Advisor Staff Paper on Risk Assessment Principles & Practices (EPA/100/B-04/001). Factors with greater variability (e.g., chemical concentrations, which can vary at the LPRSA by more than 2 orders of magnitude), influence the resulting percentile position much more than factors with more limited variability (e.g., loss of chemicals due to cooking). The staff paper notes that "selecting the mean value for the concentration input value and 95th percentile values for the others will result in a calculated exposure that is much closer to the mean of the resulting distribution than the 95th percentile (or higher), because the resulting distribution is heavily influenced by the concentration input." This statement also holds true when using the 95% UCL on the mean of the concentrations for a robust data set. Add the following to the end of the first sentence at the top of page 7-45: "consistent with guidance (USEPA 1989, 1990, 2014). Consequently, the resulting risk estimates are expected to be on the high end of the range of risks but within the range of plausible outcomes." Add the following bullet to the list on page 7-45: • 95 percent upper confidence limit on the arithmetic mean concentrations of chemicals in fish and crab tissue Revise the bullet regarding cancer slope factors from "95th percentile cancer slope factors" to • Upper bound cancer slope factors Add to end of the section: "As stated in the Cancer Guidelines and other guidance documents, within a population a portion will be at the high end of the distribution while risks to the average individual represented by the 50th percentile will be lower. This risk assessment found that the risks to the average individual (i.e., CTE scenarios) still remained above the risk range and/or the goal of protection of an HI = 1." Based on the above comment, Attachment D provides the revised Section 7.4.2 to be incorporated in the revised draft BHHRA.

No.	Page No.	Specific Comment
98	Page 7-45, Section 7.4.3 Risks to Sensitive Populations	In the last sentence, change "through the use conservative assumptions" to "through the use of health protective assumptions."
99	Page 7-48, Section 7.5 Summary of Uncertainty in BHHRA for the LPRSA	Change the statement "very conservative" to "health protective".
100	Pages 8-2 to 8-3, Section 8.1.2 Exposure Assessment	The discussion regarding EPA Region 2 needs to clarify that the values provided to the CPG are consistent with guidance. Also clarify that the RME is the basis for decisions at Superfund sites.
101	Page 8-3, Section 8.1.3 Toxicity Assessment	Remove the reference to Bowers et al. for the lead exposure. The appropriate references are the IEUBK and Lead Methodology which may incorporate the Bowers work.
102	Pages 8-4 through 8-6, Section 8.1.4 tables	Highlight the exceedance of the risk range for individual chemicals on the tables in Sections 8.1.4.1 and 8.1.4.2.
103	Page 8-8, Section 8.2 Conclusions	Indicate that the RME is the basis for the decision in appropriate bullets.
104	Page 8-10, Section 8.2 Conclusions	Last paragraph, second sentence: Change "conservative" to "health protective." Last paragraph, third sentence: Clarify that the evaluation of risks in the absence of background is consistent with guidance.
105	Pages 8-8 through 8-10, Section 8.2 Conclusions	Revise this section per comments provided on the Executive Summary (Section E.3 Conclusions).
106	Table 3-12	The report is missing Table 3-12, which is listed in the Table of Contents as "Analysis of Tissue COPCs Not Identified as Surface Water or Sediment COPCs."

No.	Page No.	Specific Comment
107	Table 5-1, chemicals using Chlordane as surrogate	Per final response to Comment 87c on the Draft BHHRA (provided in December 4, 2015 letter), a. Change the RfD for cis-Nonachlor from 1.72E-04 mg/kg-day to 1.04E-04 mg/kg-day. Change the relative potency factor (RPF) in footnote g from 2.9 to 4.8. b. Change the RfD for trans-Nonachlor from 2.51E-05 mg/kg-day to 1.55E-05 mg/kg-day. Change the RPF in footnote g from 19.9 to 32.2. Footnote g should be updated to include the November 12 and November 24, 2015, letters from STSC to Marian Olsen.
108	Table 5-2, PCBs	As noted on page 5-12 of the text, the "lowest risk and persistence" CSFs for PCBs were not used in this BHHRA. Remove these two rows from the table, or add a footnote to the table indicating the values were not used in the BHHRA.
109	Table 5-2, chemicals using Chlordane as surrogate	Per final response to Comment 87c on the Draft BHHRA (provided in December 4, 2015 letter), chlordane RPFs should apply only to the noncancer assessment and should be removed from this table. a. Change the cancer slope factor for cis-Nonachlor from 1.02E+00 $(mg/kg\text{-}day)^{-1}$ to 3.50E-01 $(mg/kg\text{-}day)^{-1}$. b. Change the cancer slope factor for Oxychlordane from 1.96E+00 $(mg/kg\text{-}day)^{-1}$ to 3.50E-01 $(mg/kg\text{-}day)^{-1}$. c. Change the cancer slope factor for trans-Nonachlor from 6.97E+00 $(mg/kg\text{-}day)^{-1}$ to 3.50E-01 $(mg/kg\text{-}day)^{-1}$. d. Change footnote g to "Value for chlordane is used as a surrogate based on structural similarity. Letters from Superfund Technical Support Center to Marian Olsen dated August 5, November 12, and November 24, 2015."
110	Tables 6-15 through 6-21, Identification of Potential COCs	Remove cis-Nonachlor, Oxychlordane, and/or trans-Nonachlor from these tables as necessary based on revised risks using the updated toxicity values. Also, add Dieldrin as a potential COC in Tables 6-15, 6-17, and 6-19 for Angler (Adult)/Crab Muscle & Hepatopancreas.
111	Table 6-21, Summary of Potential COCs By Medium and Scenario	Add an "X" to the table for PCBs (non-DLC) for RME Crab Muscle and Hepatopancreas (based on information in Table 10.7). Add gamma-Chlordane to the table with an "X" for RME Mixed Fish Diet (based on information in Table 10.7)
112	Appendix L, pages 1-1 to 1-2, Fourth Paragraph, Second Sentence	Change "The approach used for establishing background concentrations…" to "The approach used for evaluating background concentrations…"

No.	Page No.	Specific Comment
113	Appendix L, Section 3.1 Outlier Identification, Footnote 2 and Final Paragraph of Section	The footnote on page 3-1 states that BaP in surface water is not evaluated further in the background appendix. However, on page 3-3, the final paragraph in Section 3.1 is about BaP in surface water and refers to summary statistics in Table L-10. Either remove the paragraph and Table L-10, or move footnote 2 to follow this paragraph.
114	Appendix L, page 4-1, Section 4.0 Exposure Point Concentrations for Background Risk and Tables L-15 through L-19 and L-21 through L-24	Per response to Comment 218 (10/16/15) on the Draft BHHRA, the text should clarify which statistic was used as the EPC. a. At the end of the first paragraph on page 4-1, insert the sentence "The EPC is the lower of the UCL and maximum detected concentration for data sets with at least 5 detected samples; for data sets with fewer than 5 samples or 5 detects, the EPC is the maximum concentration." In Tables L-15 through L-19 and L-21 through L-24, copy footnote f from Table L-25 and insert the footnote after "Exposure Point Concentrations" in the title.

No.	Page No.	Specific Editorial Comments
1	Page 5-2, Section 5.1, Third Bullet	Change the reference for HEAST from USEPA 1997c to USEPA 1997b.
2	Page 6-35, First Full Paragraph	Change "Appendix J" to "Appendix L" where the background data were checked for outliers.
3	Page 7-18, Section 7.2.2, Third Paragraph, Third Sentence	Currently reads "Results are provided in Appendix F American eel…" Insert the word "for" after "Appendix F."
4	Page 7-24, Section 7.2.2.4, Second Paragraph, Last Sentence	Remove the phrase "…surface water, sediment, and…" because Table 7-1 just presents data for tissue.
5	Page 7-34, Section 7.3.2.3, Second Paragraph, Last Sentence	Change the reference at the end of the sentence from USEPA 2013a to USEPA 2015a.

No.	Page No.	Specific Editorial Comments
6	Page 9-17, References	For USEPA, 2014a, add the following to the end of the citation: "FAQs updated September 14, 2015."
7	Table 3-11a	The text for locations of maximum concentrations in blue crab tissue were cut off throughout the table. Please revise the row height/width accordingly.
8	Table 6-2	Values in this table should match the sitewide values presented in Table 6-8 (for RME) and Table 6-12 (for CTE), but occasionally differ because of the number of decimal places presented (e.g., 0.1 shown rather than 0.09). Please make information in these tables consistent.
9	Appendix A, List of Tables and Attachments	a. In the titles for Tables A-1, A-10, and A-19, change "Butylins" to "Butyltins" b. Add the two new attachments for data usability worksheets to this TOC list
10	Appendix A, Table A-19	In the title, change "Butylins" to "Butyltins"
11	Appendix L, page 2-1, Table L-1	Correct the number of fish tissue samples from above Dundee Dam. Change "50 fillet samples" to "47 fillet samples" based on the information in later tables (e.g., Table L-4).
12	Appendix L, Table L-5	a. There should only be 3 samples listed for Smallmouth Bass. Remove the repeated samples. b. Insert the missing Northern Pike sample.
13	Appendix L, Table L-9	Footnote j (about "Blue crab – muscle only") is missing. Please add.
14	Appendix L, Table L-18	Correct the FOD for Hexachlorobenzene from 3:6 to 3:3.

No.	Page No.	Specific Editorial Comments
15	Appendix L, Table L-20	Remove "Largemouth &" from column header for Smallmouth Bass. Correct information in footnotes a through e as follows: a. American eel EPC selected in Table L-16 (not L-13) b. Channel catfish EPC selected in Table L-17 (not L-14) c. Common carp EPC selected in Table L-19 (not L-16) d. Smallmouth bass EPC selected in Table L-18 (not L-15, and remove Largemouth bass from footnote) e. White perch EPC selected in Table L-15 (not L-12)

Attachment A
Revised text for Section ES.3 Conclusions and 8.2 Conclusions

The conclusions of the BHHRA are summarized below:

Fish and Crab

The predominant source of potential risk to human health is from consumption of LPRSA fish and crab. At RME exposure levels, which represent an upper bound ingestion rate by definition, the potential cancer risks and noncancer hazards to recreational anglers who are assumed toregularly consume their catch (i.e., eat approximately 56 LPRSA fish meals per year or approximately 30 meals per year of 6 crabs per meal) exceed the values used by USEPA for determining whether a site poses unacceptable risk. RME cancer risks are up to 40 times higher than the NCP cancer risk range (up to 4×10^{-3} for fish ingestion and 1×10^{-3} for crab ingestion). RME noncancer hazards are up to 200 times higher than the goal of protection of an HI = 1 (total HIs up to 193 for fish ingestion and 50 for crab ingestion).

At average (CTE) exposure levels (about six fish meals or four crab meals per year, accounting for cooking loss, and assuming fewer years of eating LPRSA fish/crab), the potential cancer risks to recreational anglers who consume LPRSA fish or crab are within the NCP risk range of 1×10^{-6} to 1×10^{-4}. Potential CTE noncancer hazards are six to seven-fold lower than the RME hazards, but still exceed an HI of 1 (total HIs up to 15 for fish ingestion and 8 for crab ingestion).

The dominant potential COCs for the fish and crab consumption scenarios are TCDD-TEQ and PCBs, with mercury and pesticides also contributing to cumulative RME and CTE risks/hazards. The percent contributions of key potential COCs (presented in Appendix J) are summarized below for the RME scenario, with ranges presented where there are differences by receptor and PCB toxicity approach.

Fish consumption

- Cancer risk – TCDD-TEQ contributes approximately 67% to 76% (maximum risk of 3×10^{-3}), PCBs contribute approximately 20% to 30% (maximum risk of 7×10^{-4}), and pesticides contribute less than 5% to total potential cancer risk (maximum risk among pesticides of 7×10^{-5}, for dieldrin).
- Noncancer hazard – TCDD-TEQ contributes approximately 53% to 58% (maximum HQ of 102), PCBs contribute approximately 20% to 30% (maximum HQ of 85), and methyl mercury and pesticides each contribute about 1% (maximum HQ of 2 for methyl mercury) to total potential noncancer hazard. Potential health effects associated with the RME noncancer hazards exceeding an HI = 1 are: developmental and reproductive effects (TCDD-TEQ and dioxin-like PCBs); eye, nails and immune effects (PCBs); and neurological effects (methyl mercury).

Crab consumption

- Cancer risk – TCDD-TEQ contributes approximately 76% to 89% (maximum risk of 1×10^{-3}), PCBs contribute approximately 7% to 20% (maximum risk of 3×10^{-4}), and pesticides contribute less than 4% to total potential cancer risk(maximum risk among pesticides of 2×10^{-5}, for dieldrin).
- Noncancer hazard – TCDD-TEQ contributes approximately 70% to 80% (maximum HQ of 35), PCBs contribute approximately 17% to 26% (maximum HQ of 13), and methyl mercury and pesticides each contribute about 1% (maximum HQ of 0.6 for methyl mercury) tototal potential noncancer hazard. Potential health effects associated with the RME noncancer hazards exceeding an HI = 1 are: developmental and reproductive effects (TCDD-TEQ and dioxin-like PCBs); eye, nails and immune effects (PCBs); and neurological effects (methyl mercury).

For the LPRSA fish and crab risks/hazards, 2,3,7,8-TCDD contributes the majority of total TEQ risk/hazard (approximately 78% to 80%). Approximately 16% to 17% of total TEQ is attributable to PCB-TEQ, and approximately 3% to 5% is attributable to the other TCDD-TEQ congeners. There is a greater certainty in estimates of risk/hazard from 2,3,7,8-TCDD and its contribution to total risk/hazard than for the other dioxin-like compounds (USEPA 2010d, 2013e).

The species and tissue types that comprise the fish/crab diet influence risk, as some species and tissue types exhibit greater tissue burdens of bioaccumulative chemicals than others. LPRSA fillet data were collected and analyzed for the following species: American eel, channel catfish, common carp, bass (largemouth and smallmouth), northern pike, white catfish, white perch, and white sucker. Risks estimated using the LPRSA fillet data for any combination of these species still exceed the NCP cancer risk range under the RME scenario and the noncancer goal of protection under both the RME and CTE scenarios. For example, an alternate mixed fish diet that excludes carp could pose potential risks/hazards that are approximately three-fold lower than a mixed diet that includes common carp, but still exceed the NCP cancer risk range and noncancer goal of protection. Similarly,a crab muscle-only diet (i.e., removing the hepatopancreas prior to cooking) could pose risks/hazards that are approximately five to six-fold lower than a diet of crab cooked whole and consumed with its cooking juices, but such a crab muscle-only diet would still exceed a noncancer HI of 1 for young children (HI=9). Not consuming the cooking juices or pan drippings could reduce risk from both fish and crab consumption.

Sediment and Surface Water

Potential RME cancer risks from direct contact exposures to accessible surface sediment and surface water in the LPRSA are significantly lower than fish and crab ingestion, and are within the NCP risk range. Recreational exposure to accessible surface sediment and surface water during boating, wading, fishing, or swimming in the LPRSA and worker exposures to accessible surface sediment do not pose unacceptable cancer risks or noncancer hazards under the RME or CTE scenarios, with the exception of surface sediment exposure in the RM 6-9 area. An analysis of direct contact exposure to accessible surface sediment by 3-mile river segments indicates that only RM 6-9, and specifically the East Bank, poses potential RME and CTE noncancer hazards in excess of an HI of 1 (maximum HI of 5 for RME and 2 for CTE), due primarily to TCDD-TEQ, which contributes over 90% of noncancer hazards. The potential RME and CTE sediment contact cancer risks in all river segments are within the NCP risk range (maximum risk of 7×10^{-5} for the East Bank of RM 6-9).

Background

Background levels of several potential COCs pose fish and crab consumption risks that exceed the upper end of the NCP risk range of 1×10^{-6} to 1×10^{-4} for cancer and the goal of protection of an HI equal to 1 for noncancer.

- Fish: The potential cancer risk from PCBs in background (UPR) fish is above the risk range (~3×10^{-4} to 5×10^{-4}) and at the upper end of the risk range for dieldrin (~8×10^{-5}). The potential noncancer hazard from PCBs in background fish exceeds an HI of 1 (9 to 22), as does methyl mercury (HI of 3). The background risks/hazards for pesticides and methyl mercury in fish are about the same as LPRSA risks/hazards. Background PCB risks/hazards are about one third of corresponding LPRSA risks/hazards for consumption of the mixed fish diet. Background risks/hazards for TCDD-TEQ are only about 2% of the corresponding LPRSA risks/hazards for consumption of the mixed fish diet.
- Crab: The potential background cancer risks and noncancer hazards from consuming Jamaica Bay crab (muscle and hepatopancreas) are approximately 30% to 70% of corresponding LPRSA risks/hazards for PCBs, methyl mercury, heptachlor epoxide, and dieldrin, and approximately 8% of LPRSA risks/hazards for TCDD-TEQ.

Thus, upstream and regional levels of several potential COCs, including PCBs, pesticides, and mercury, are elevated and may contribute to levels observed in the LPRSA and to risks estimated for LPRSA receptors. The distinguishing potential COC for the LPRSA when compared to other regional waterbodies is TCDD-TEQ.

As with all risk assessments, assumptions have been made about variables and processes that are not fully known, such as human behavior, chemical toxicity, or environmental concentrations. While the use of assumptions leads to uncertainty, it is important to note thatthe assumptions and approaches used in this BHHRA are health protective.

Attachment B
Revised text for Section 7.2.3.2 Oral Bioavailability

7.2.3.2 Oral Bioavailability

As noted above, 100% oral bioavailability of chemicals in aged sediment is unlikely and the use of this assumption has likely resulted in an overestimation of potential risks from incidental ingestion of LPRSA sediments; although this pathway did not pose a risk above the risk range or the goal of protection of an HI = 1. COPCs where the default bioavailability assumption may be overestimated include TCDD-TEQ, PCBs, PAHs, and arsenic, as summarized below. EPA has only developed an oral bioavailability factor for use at Superfund sites for arsenic. This bioavailability factor was used in the calculations of risks and hazards.

TCDD-TEQ
EPA's 2015 guidance on relative bioavailability of TCDD-TEQ identified nine studies that collected data on soil RBA based on bioassays conducted in guinea pigs (McConnell et al., 1984; Umbreit et al., 1986; Wendling et al., 1989), rabbits (Bonaccorsi et al., 1984); rats (Budinsky et al., 2008; Finley et al., 2009; Lucier et al., 1986; Shu et al., 1988) or swine (Budinsky et al., 2008; Wittsiepe et al., 2007). These studies used various experimental designs for dosing animals, metrics for estimating bioavailability, and data reduction methods for calculating soil RBA. The 2015 document indicated that the extent to which variations in experimental design affects RBA estimates has not been rigorously evaluated. Only one study compared RBA estimates for the same test materials in more than one assay; the outcome was dissimilar estimates of RBA for 2 soils based on a single dose rat bioassay and a multiple dose swine assay (Budinsky et al., 2008).

The 2015, EPA document titled "**Soil Dioxin Relative Bioavailability Assay Evaluation Framework"** methods for estimating RBA of PCDD/F in soil and concluded these methods are in the early development phase". The report indicates the methodology for assaying PCDD/F RBA in soils is evolving, greater experience with various experimental designs is likely to prompt modifications to the requirements identified in the report. The USEPA did not recommend a default oral absorption factor for TCDD-TEQ so that a RBA factor could not be used in this assessment.

PCBs and PAHs
Over the years, there have been a number of studies regarding the bioavailability of PCBs and PAHs in soils developed by various researchers. Research is on-going and currently EPA does not have a recommended value for bioavailability in soils for these compounds.

Arsenic (inorganic)
The arsenic dose-response values are based on drinking water studies, and in the absence of site specific data, it has typically been assumed that absorption of arsenic from soil or sediment is the same as absorption from drinking water. However, recent in-vivo bioavailability studies show that this is not the case for arsenic, and that the bioavailability of arsenic in soil is less than the bioavailability of arsenic dissolved in drinking water (USEPA 2012c). Therefore, the assumption of 100% RBA will result in an overestimate of risk via the oral pathway. USEPA derived an RBA of 60% for soils based on a review of over 100 arsenic RBA estimates (USEPA 2012c), and this RBA was used in the BHHRA.

Attachment C
Revised text for Section 7.3 Toxicity Assessment

The purpose of the toxicity assessment is to identify the types of adverse health effects a chemical may potentially cause and to define the relationship between the dose of a chemical and the likelihood or magnitude of an adverse effect (response). USEPA has published guidelines on the development of toxicity values for use in risk assessments, including the application of uncertainty factors for derivation of non-cancer toxicity values e.g., reference doses and reference concentrations and models for estimating cancer slope factors (CSFs for oral and Inhalation Unit Risks for inhalation exposures) (USEPA 2002f, 2005b, 2012f) [http://www.epa.gov/risk/risk-assessment-guidelines]. Risk assessment methodologies typically divide potential health effects of concern into two general categories: effects with a threshold (noncarcinogenic) and potential carcinogens. The Cancer Guidelines (USEPA 2005b) highlight the "difficulty of empirically distinguishing a true threshold from a dose-response that is non-linear at low doses" commonly referred to as a threshold for carcinogens. Toxicity assessments for both of these types of effects share many of the same sources of uncertainty. EPA uses upper bound estimates that are generally considered to be a health-protective approach for covering the risk to susceptible individuals, although the calculation of upper bounds is not based on susceptibility data. Similarly, exposure during some lifestages can contribute more or less to the total lifetime risk than do similar exposures at other times. The dose-response assessment characterizes, to the extent possible, the extent of these variations (USEPA 2005).

Humans are typically exposed to chemicals in the environment at levels much lower than those tested in animals. For certain chemicals, these low doses may be detoxified or rendered inactive by the myriad of protective mechanisms that are present in humans (Ames et al. 1987) and which may not function at the high dose levels used in animal experiments. Moreover, as noted by USEPA (1993d) "in the case of systemic toxicity, however, organic homeostatic, compensating, and adaptive mechanisms exist that must be overcome before a toxic endpoint is manifested." Therefore, some limitations exist in using the results of these animal studies to accurately predict a dose-response relationship in humans (USEPA 1989b). The Cancer Guidelines (USEPA 2005b) discuss various whole-animal test systems currently used or being developed for evaluating potential carcinogenicity to humans. Cancer studies involving chronic exposure for most of the lifespan of an animal are generally accepted for evaluation of tumor effects (Tomatis et al., 1989; Rall, 1991; Allen et al., 1988; Ames and Gold, 1990). Other studies that use other designs are useful for observing formation of preneoplastic lesions or tumors or investigating specific modes of action. Their applicability is determined on a case-by-case basis based on effects observed in animal models for humans. This information is used in a variety of ways, from determining the role of metabolism in toxicity, to assessing whether homologous activity would be expected across species; to determining whether or not a threshold is likely to exist for the response. This information is weighed by EPA in the development of toxicity values in light of the known heterogeneity of the human population versus the relatively inbred status of laboratory animals used in toxicity testing studies and housed under carefully controlled environmental conditions (USEPA 2005b).

Despite these uncertainties, and with the goal of being protective of human health, USEPA assumes that the results of animal toxicity studies are predictive of potential toxicity in humans. Moreover, based on the assumption that humans are more sensitive to chemicals than laboratory animals, USEPA incorporates health protective assumptions and UFs when deriving numerical toxicity values from laboratory studies, as discussed below. However, USEPA explicitly recognizes these extrapolations from high doses to low doses and from animal studies to predict responses in humans as uncertainties in the risk assessment process (USEPA 1989b).

In some cases, data from human exposure to chemicals are used to develop dose-response values. However, these data also have uncertainties because often it is not possible to determine from human exposure studies whether one or more chemicals are responsible for the observed effects, and in general it is even more difficult to determine precise exposure levels (USEPA 1989b). Consistent with the 2005 Cancer Guidelines (USEPA, 2005b) conclusions regarding the strength of the evidence for positive or negative associations observed from human epidemiological evidence, as well as evidence supporting judgments of causality, are described as part of the assessment. In assessing the human data within the overall weight of evidence approach, determining the strength of the

epidemiologic evidence clearly identifies the degree to which the observed associations may be explained by other factors, including bias or confounding. Moreover, where effects are observed in humans, they generally occur at high exposure levels (often in industrial settings), and it is difficult to predict potential human responses at the much lower dose levels that occur in environmental exposure scenarios (USEPA 1989b). The Cancer Guidelines describe extrapolation approaches that consider the understanding of the chemical's mode of action at each tumor site, and the extent of inter-individual variation with greater variation spreading the response over a wider range of doses.

7.3.1 Evaluation of Noncarcinogenic Dose-Response

For many chemicals, animal studies provide the only reliable information on which to base an estimate of adverse human health effects. Of the 49 COPCs evaluated quantitatively in the BHHRA, 42 have oral reference doses, of which 29 are based on animal studies and 13 are based on human studies. One of the major default assumptions in EPA's risk assessment guidelines (USEPA 2002) is that animal data are relevant for humans (e.g., USEPA, 1991, 1996, 1998c, 2002). Such defaults are used in the absence of experimental data that can provide direct information on the relevance of animal data. Several types of information are considered when determining the importance of effects observed in animal models for humans. As described above, extrapolation from animals to humans introduces uncertainty into the risk characterization; where human studies are available, uncertainty is reduced. The chemical-specific toxicity information is weighed in light of the known heterogeneity of the human population versus the relatively inbred status of laboratory animals used in toxicity testing studies and housed under carefully controlled environmental conditions to develop appropriate toxicity values. If a chemical's fate and the mechanisms by which it causes adverse effects are known in both animals and humans, uncertainty is reduced. When the fate and mechanism for the chemical are unknown, uncertainty increases.

The procedures used to extrapolate from animals to humans involve health protective assumptions and incorporate Uncertainty Factors (UFs) such that overestimation of effects in humans is more likely than underestimation. When data are available from several species, the lowest dose that elicits effects in the most sensitive species is used for the calculation of the RfD. To this dose are applied UFs, generally ranging from 1 to 10 each to account for intraspecies variability, interspecies variability, study duration, and/or extrapolation of a low effect level to a no effect level. The combined UFs for the COPCs evaluated in this risk assessment range from 1 (manganese, diet) to 3000 (cobalt, thallium, fluoranthene (surrogate for C2-benzanthracenes), and naphthalene). For the PPRTV appendix screening RfD for C9-C18 hydrocarbons, the combined UF is 10,000. As previously noted in Section 5.2, the PPRTV appendix RfD for C9-C18 hydrocarbons is associated with greater uncertainty and intended for screening purposes only. USEPA (2002f) recommends limiting the total combined UF for a chemical to 3000.

Nevertheless, because the fate of a chemical can differ in animals and humans, it is possible that animal experiments will not reveal an adverse effect that would manifest itself in humans. This can result in an underestimation of the effects in humans. The opposite may also be true: effects observed in animals may not be observed in humans, resulting in a potential overestimation of adverse human health effects.

7.3.2 Evaluation of Carcinogenic Dose-Response

Uncertainties exist in estimating dose-response relationships for potential carcinogens. These are due to experimental and epidemiologic variability, as well as uncertainty in extrapolating both from animals to humans and from high to low doses. Three major issues affect the toxicity assessments used to estimate potential excess lifetime cancer risks: (1) the selection of a study (i.e., data set, animal species, matrix the chemical is administered in) upon which to base the calculations, (2) the conversion of the animal dose used to an equivalent human dose, and (3) the mathematical model used to extrapolate from experimental observations at high doses to the lower doses potentially encountered in the environment. Of the 49 COPCs evaluated quantitatively in the BHHRA, 30 are classified by USEPA as potentially carcinogenic to some degree via the oral route of exposure. The IRIS Agenda indicates that the program has deferred completion of the dioxin cancer assessment at this time.

7.3.2.1 Study Selection

USEPA's process for selecting the studies for use in developing CSFs involves internal and external peer review, as well as public comment and review. As part of scoping, the review process for chemical assessment, a public meeting is held to obtain input from the scientific community and the general public. Study selection involves the identification of a data set (experimental species and specific study) that provides sufficient, well-documented dose-response information to enable the derivation of a valid CSF. Human epidemiological data are preferable to animal data, although adequatehuman data sets are more limited. Therefore, it is often necessary to develop dose-response information from a laboratory species, ideally one that biologically resembles humans (e.g., with respect to metabolism, physiology, and toxicokinetics), and where the route of administration is similar to the expected mode of human exposure (e.g., inhalation and ingestion).

The dose-response assessment for a chemical is based on the mode(s) of action for each tumor type. Because a chemical may induce multiple tumor types, the dose-response assessment includes an analysis of all tumor types, followed by an overall synthesis of information that includes a characterization of the risk estimates across tumor types, the strength of the mode of action information of each tumor type, and the anticipated relevance of each tumor type to humans, including susceptible populations and lifestages (e.g., early childhood).

The current study selection criteria are designed to be health protective as described above. Health protective means that estimates, while uncertain, are more likely to overstate than understate hazard and/or risk. The oral CSF for only three COPCs (inorganic arsenic, benzene, and trichloroethene) are based on human epidemiological studies. None of these chemicals contribute significantly to cumulative site risk. The oral cancer slope factors for all of the other COPCs evaluated in the BHHRA are based on animal studies. Most of these COPCs are classified as "B2" which is defined as probably carcinogenic to humans based on evidence in animals, but with little or no human data, under the 1986 cancer classification approach (USEPA 1986). One COPC (heptachlor epoxide) is classified under the newer (2005a) scheme as "Likely to be Carcinogenic to Humans" for the oral route.

USEPA's IRIS database does not currently provide a cancer classification for TCDD, and indicates that "the IRIS Agenda has deferred completion of the dioxin cancer assessment at this time." Due to the lack of a final peer reviewed and consensus-based CSF for TCDD, a Tier 3 value was used, as discussed in Section 5.5.4. The CSF of 150,000 per mg/kg-day (USEPA 1996, 1997b) was directed for use in the BHHRA by USEPA Region 2 based on USEPA 1996. The uncertainty associated with the selected Tier 3 CSF for TCDD, including a summary of other published CSFs, is discussed in Section 7.3.6.1.

PCBs have been demonstrated to produce tumors in animals, and the tiers of CSFs for PCBs listed in IRIS are based on a rat diet study (USEPA 1996, 2015a). Several studies have interpreted human epidemiological data for workers as negative (Shields 2006, Golden et al. 2003, Golden and Kimbrough 2009). A recent review of non-occupational case-control epidemiological studies linked PCBs with non-Hodgkin Lymphoma in humans (Kramer et al. 2012). IRIS identifies human carcinogenicity data for PCBs to be "inadequate but suggestive"; however, USEPA has determined the weight of evidence to be sufficient to classify PCBs as a probable human carcinogen (B2) (USEPA 2015a). The International Agency for Research on Cancer (IARC) recently concluded there is sufficient human evidence and classified PCBs as Group 1 (carcinogenic to humans) (IARC 2015).

Further details on the data available on the potentially carcinogenic COPCs evaluated in the BHHRA may be found in the IRIS chemical files, PPRTV files and other documents supporting the development of the CSFs used in this assessment. Depending on the database of information availableon each chemical, potential cancer risks may be over- or underestimated, although the intent of the development process, as described above, is that the resulting CSF represents the upper bound on the average risk in a population (USEPA 2005b).

7.3.2.2 Interspecies Dose Conversion

The USEPA derivation of human equivalent doses by conversion of doses administered to experimental animals requires the assumption that humans and animals are equally sensitive to the toxic effects of a substance, if the same dose per unit body surface area is absorbed by each species, and the mechanism of toxicity is the same. The interspecies evaluation includes evaluation of biological markers of effects in biological systems or samples; identifies doses at which elements of the carcinogenic process are operating; aids in interspecies extrapolations when data are available from both experimental animal and human cells; and under certain circumstances, provides insights into the possible shape of the dose-response curve below levels where tumor incidences are observed (USEPA 2005b). The Cancer Guidelines (USEPA 2005b) states that interspecies dose conversions that result in slope factors generally represent an upper bound on the average risk in a population or the risk for a randomly selected individual but not the risk for a highly susceptible individual or group. Some individuals face a higher risk and some face a lower risk. The use of upper bounds generally is considered to be a health-protective approach for covering the risk to susceptible individuals, although the calculation of upper bounds is not based on susceptibility data.

Further assumptions for dose conversions involve standardized scaling factors to account for differences between humans and experimental animals with respect to life span, body size, breathing rates, and other physiological parameters. In addition, evaluation of risks associated with one route of administration (e.g., inhalation) when tests in animals involve a different route (e.g., ingestion) requires additional assumptions with corresponding additional uncertainties. Although USEPA has formally changed its default position for scaling animal data to humans from a per surface area basis to a per body weight basis (USEPA 1992d, 2005b), changes to existing CSF will be made when the USEPA updates toxicity values through the IRIS process. It is noted that USEPA's 1996 cancer assessment for PCBs included a body weight to the ¾ power extrapolation (USEPA 1996).

7.3.2.3 High-to-Low Dose Extrapolation

The concentration of chemicals to which humans are potentially exposed in the environment is usually lower than the levels used in the studies from which dose-response relationships are developed. Estimating potential health effects, therefore, requires the use of models that allow extrapolation of health effects from high experimental doses in animals to low environmental doses. Thus, the use of a model for dose extrapolation introduces uncertainty in the dose-response estimate. These models are generally statistical in character and are health protective meaning that estimates, while uncertain, are more likely to overstate than understate hazard and/or risk. The NRC (1994) reaffirmed the use of default options as "a reasonable way to cope with uncertainty about the choice of appropriate models or theory" (p. 104).

Many of the USEPA CSFs listed in IRIS are derived using the upper 95% confidence limit of the slope predicted by models such as the Linearized Multistage (LMS) used to extrapolate to low dose risk from high dose experimental data. USEPA's 2005 Cancer Guidelines lays out an approach for evaluating data and models that are used to extrapolate from high dose to low dose. The Cancer Guidelines states "that the upper-bound estimate generated by the LMS model leads to a plausible upper limit to the risk that is consistent with some of the proposed mechanisms of carcinogenesis". The true risk, however, is unknown and may be as low as zero. The LMS model assumes linearity between the lowest dose that produced an effect and zero dose. According to USEPA (1989b), "Because the slope factor is often an upper 95th percentile confidence limit of the probability of response based on experimental animal data used in the multistage model, the carcinogenic risk estimate will generally be an upper-bound estimate. This means that USEPA is reasonably confident that the "true risk" will not exceed the risk estimate derived through use of this model and is likely to be less than that predicted." Consequently, the assumption that there is some probability of harm to human health at any level of exposure is very conservative and is expected to result in overestimates of risk, especially when coupled with the use of an upper bound estimate of cancer potency.

USEPA's current carcinogen risk assessment guidelines (USEPA 2005b) emphasizes mode of action data, and recognizes that some carcinogens may act in a nonlinear fashion. Therefore, it is recognized that some carcinogens may have a nonlinear dose below which effects would not beseen. For example, a

nonlinear dose for carcinogenic activity has been demonstrated for chloroform and was used as the basis for USEPA's development of dose-response values for chloroform (USEPA 2015a). However, chloroform was not a COC at the LPRSA.

USEPA's IRIS program utilizes a systematic literature review in the development of individual chemical files (USEPA 2015a). The approach is outlined on the IRIS webpage including the IRIS Agenda for chemicals being reassessed, process for developing an IRIS assessment include systematic review, peer-review procedures, and issuance of the final document. The IRIS Webpage www.epa.gov/iris provides details on the IRIS process. For example, USEPA is updating the chemical assessment for inorganic arsenic using these procedures and will consider comments based on the 2011 Science Advisory Board (SAB) recommendations [available at: http://yosemite.epa.gov/sab/sabproduct.nsf/fedrgstr_activities/9FCEE4E20ABD6EB48525784600791AC2/$File/EPA-SAB-11-003-unsigned.pdf].

Much of the knowledge about the health effects of TCDD and other DLCs in humans comes from studies of relatively highly exposed populations in the workplace, from explosions such as that in Seveso, Italy, and from U.S. military exposed by spraying Agent Orange during the Vietnam War. The potential adverse effects of TCDD and DLCs from long-term, low-level exposures to the general public are areas of ongoing investigation. To complicate matters, experimental data from animal bioassays similarly reflect effects associated with much higher exposure to TCDD and related compounds than would be expected in the general environment. Estimating risks from the doses employed in the studies to typical human exposure levelsrequires making assumptions about the point at which adverse effects are considered to occur (point of departure), methods for modeling the effects at doses below this point (linear vs. nonlinear extrapolation), the relationship of the doses in animals to the concentrations in humans (scaling vs. physiologicaly based pharmacokinetic modeling), and the relationship of the observed effects to mixtures of these compounds in the environment, among others.The IRIS Agenda indicates that the program has deferred completion of the dioxin cancer assessment at this time.

7.3.3 Uncertainty in TEF Approach

Application of the TEF approach to the human health risk assessment of dioxin-like congeners (DLCs) carries with it a number of uncertainties. These have been discussed in the literature and guidance (Haws et al. 2006, NAS 2006, USEPA 2003c, 2010d, van den Berg et al. 1998, 2006), and are summarized in USEPA (2010d), which recommends the 2005 WHO consensus TEFs, but does not address specific risk assessment applications of TEFs. To be considered a DLC, a chemical must:

- Show a structural relationship to the PCDDs and PCDFs,
- Bind to the Ah receptor (AhR),
- Elicit AhR-mediated biochemical and toxic responses, and
- Be persistent and accumulate in the food chain (Van den Berg et al. 2006).

Expert judgment and a consensus process of scientific expert panels to develop the WHO 2005 TEFs (van den Berg et al. 2006), including evaluation of information from the Haws et al. (2006) database of laboratory studies in which the relative potency of a test compound was compared to a reference compound, usually 2,3,7,8-TCDD. As described in USEPA 2010, after evaluating the empirical data on TCDD and some DLCs, WHO reconfirmed that the combined effects of these compounds generally are consistent with dose additivity, a key underlying assumption of the TEF methodology (van den Berg et al., 2006). The hierarchy for the TEF derivation preferred the use of in vivo studies (studies done in animals), but included in vitro studies (studies done on enzymes, cells, tissues or body fluids, but outside the whole animal) if in vivo data were not available. However, the in vivo studies in the database were generally short-term studies of noncancer endpoints, the assumption being that the relative potency measured for a test compound and the reference compound for a short-term noncancer endpoint would also be the same relative potency that would be observed for a two-year cancer bioassay. This was also the basic assumption for the in vitro results, i.e., that the comparative potency measured in the in vitro study is also predictive of potency of tumor induction. Other

toxicological data considered for these comparisons of toxic potency included structure-activity relationships and are based on the following classes of measure: biochemical changes, toxicity, and carcinogenicity. Uncertainty is introduced each time the assumption of similar mechanism is employed, specifically in the assumptions that:

- 2,3,7,8-TCDD induces tumors in laboratory animals and, therefore, will also do the same in humans.
- A compound that can elicit a similar toxicological response in laboratory animals as 2,3,7,8-TCDD based on the measurement of a single toxicological endpoint will also act by the same mechanism of action as 2,3,7,8-TCDD and will also induce tumors in animals and, therefore, humans.
- A compound that can elicit a similar toxicological response in an in vitro assay as 2,3,7,8-TCDD will act by the same mechanism of action as 2,3,7,8-TCDD in vivo and will also induce tumors in animals and, therefore, humans.

A number of uncertainties regarding the TEF approach are presented in USEPA (2010d). In summary, while point estimate TEFs have been identified and were used to estimate risk for DLCs, they are known to be variable and uncertain (Van den Berg et al. 2006, USEPA 2010d).

The uncertainty in TEQ estimates and in the TEF methodology accounts for only some of the overall uncertainty in a risk assessment of DLCs. There is also uncertainty associated with assessing exposures to environmental mixtures of TCDD and DLCs and with quantitatively linking health effects to the TCDD and DLC exposures (USEPA 2010d). As noted in Section 5.5.2, USEPA (2013e) recommends that risk assessors identify the fraction of the total TEQ attributable to 2,3,7,8-TCDD (for which uncertainty is "relatively low") and the fraction attributable to DLCs (for which uncertainty is "somewhat higher"). The following table presents the DLC cancer risks for the RME adult/child angler consuming the RME mixed fish diet and a crab muscle and hepatopancreas diet, and the fraction attributable to each of three principal TEQ components.

Add-in Table

As shown above, the majority (78% to 80%) of the Total TEQ risk from fish/crab consumption is attributable to 2,3,7,8-TCDD, for which uncertainty is relatively low. A small fraction (3 to 5%) of the Total TEQ risk is attributable to other PCDD and PCDF congeners. The remainder (16% to 17%) of the Total TEQ risk is attributable to PCB-TEQ, for which USEPA states that uncertainty is somewhat higher.

The following table presents the DLC noncancer hazards for the RME young child angler consuming the RME mixed fish diet and a crab muscle and hepatopancreas diet. As shown below, the majority (78% to 80%) of the Total TEQ hazard from fish/crab consumption is attributable to 2,3,7,8-TCDD, a small fraction (3 to 5%) is attributable to other PCDD and PCDF congeners, and the remainder (16% to 17%) is attributable to PCB-TEQ.

Add-in Table

The application of TEFs to the evaluation of PCB risk/hazard is an area of uncertainty. The TEFs for PCB DLCs have been developed based on a database of laboratory studies in which the relative potency of a test compound was compared to a reference compound, usually 2,3,7,8-TCDD. However, there is uncertainty in the assumption that a subset of PCB congeners exerts toxicity in a manner similar to that of 2,3,7,8-TCDD. As described in USEPA 2010, "In its review, NAS supported the use of the TEF approach (NAS, 2006, p. 8), stating that even with the inherent uncertainties, the committee concludes that the TEF methodology provides a reasonable, scientifically justifiable, and widely accepted method to estimate the relative potency of DLCs."

The application of the current dioxin TEF scheme introduces uncertainty into risk assessment of PCBs. PCBs that are approximate stereoisomers of dioxin/furan Ah receptor agonists bind the receptor much more weakly than strong Ah receptor agonists such as dioxins and furans. Even with the most

favorable chlorination pattern, the affinity of PCBs for the Ah receptor is not nearly that of potent dioxin/furans. Only a handful of Ah receptor agonists have been tested for human Ah receptor affinity even though marked species differences have been demonstrated.

In addition to the specific TEF derivation and applicability considerations, it must be noted that the PCB mixtures upon which the PCB high risk and persistence CSF is based may have included dioxin-like PCBs indicating "lot-to-lot differences highlight the importance of characterizing and reporting mixture composition (Cogliano, 1998). Conducting separate TEF-based risk calculations was performed consistent with guidance (USEPA 1996). The uncertainty associated with applying DLC TEFs to PCBs was addressed in part by evaluating PCB cancer risk two ways in the BHHRA: 1) as total PCBs using the CSFs for PCB mixtures, and 2) as the sum of PCB-TEQ using the DLC TEFs and non-DLCs using the CSFs for PCB mixtures. The following tables present the RME mixed fish diet and crab muscle and hepatopancreas consumption risks/hazards for the two approaches, includingthe percent increase in risk/hazard from using the TEQ/non-DLC approach. The risk/hazard posed by TCDD-TEQ is included for informational Purposes.

Include Table

As shown above, the approach of summing PCB-TEQ and PCBs (non-DLC) results in a 72% increase in fish consumption cancer risk and a 23% increase in noncancer hazard relative to the risk/hazard posed by evaluating total PCBs alone. These findings suggest there is enrichment of dioxin-like congeners in the fish tissue.

Include Table

As shown above, the approach of summing PCB-TEQ and PCBs (non-DLC) results in a more than three-fold increase in crab consumption cancer risk and a 75% increase in noncancer hazard relative to the risk/hazard posed by evaluating total PCBs alone. The findings suggest there is enrichment of dioxin-like congeners in the LPRSA biota, especially in crab muscle and hepatopancreas tissue (although this has a marginal impact on cumulative site risks/hazards).

As previously noted in Section 5.5.2, the approach of summing PCB-TEQ and non-DLCs has the potential to overestimate PCB risk/hazard, while the approach of evaluating total PCBs using the IRIS CSFs and Aroclor 1254 RfD may underestimate risk/hazard. It isimportant to note that the PCB Aroclor mixtures upon which the PCB high risk and persistence CSF is based included dioxin-like PCBs (Cogliano 1998; Mayes et al. 1998). Therefore, the study results represent the summed toxicities of all of the congeners in each mixture, including dioxin-like and non-dioxin-like activities. The high risk and persistent CSFs of 1 and 2 (mg/kg-day)–1 listed on IRIS should therefore be protective of exposures to the amount of TEQ from the 12 "dioxin-like" congeners present in the Aroclor 1254 bioassay test material. The amount of TEQ in the Aroclor 1254 cancer bioassay mixture has been reported to be 46 to 48 mg-TEQ/kg-PCBs (Brown et al., 1996 as cited in Cogliano 1998, Mayes et al. 1998). The TEQ amount reported in the 1998 study was calculated using earlier TEFs (i.e., Ahlborg et al. 1994). Using current WHO 2005 TEFs, the TEQ amount in the Aroclor 1254 bioassay material has been reported to be approximately 21 mg-TEQ/kg-PCBs (Brown et al. 2007). The Tables above, however, indicate that individually the cancer risks of non-dioxin-like PCBs and DLC PCB still represent a cancer risk above the risk range and a non-cancer HI greater than the goal of protection of an HI = 1.

As shown in the figure below, TEQ levels in the LPRSA RME mixed fish diet species are all below this range, suggesting the PCB CSF is adequately protective for evaluating potential cancer risks of PCBs in LPRSA fish, and an additive analysis of PCB-TEQ risks overstates PCB cancer risks. The levels in LPRSA crab muscle and hepatopancreas slightly exceed the TEQ level adjusted for 2005 WHO TEFs, suggesting that consideration of potential TEQ risk may be warranted for crab.

Include figure

However, as previously noted, there remains the issue of potential overestimation of cancer risk and noncancer hazard from summing PCB-TEQ and non-DLCs without adjusting for the lower toxicity of the non-DLC fraction, as well as the aforementioned uncertainties associated with the TEFs for PCBs. Subtraction of the mass of dioxin-like congeners from the mass of total PCBs does not resolve double-counting of toxicity, as the CSF and RfD used to evaluate the non-dioxin-like fraction are based on all congeners in the Aroclor mixture. This includes the dioxin-like congeners, which contribute significantly to the cancer and non-cancer toxicities of the mixture. Thus, the use of unadjusted total PCB toxicity values (e.g., upper-bound PCB CSF of 2 mg/kg-day-1 or Aroclor 1254 RfD of 2E-05 mg/kg-day) to evaluate the non-dioxin-like PCBs overestimates the risk/hazard of this fraction, although the degree of overestimation is uncertain. However, the Tables above indicate that individually the cancer risks of non-dioxin-like PCBs and DLC PCB still represent a cancer risk above the risk range and a non-cancer HI greater than the goal of protection of an HI = 1.

Attachment D
Revised Text for Section 7.4.2 Combination of Several Upper-Bound Assumptions

Generally, the goal of a risk assessment is to estimate risk to the RME individual. As previously discussed in Section 4, this should be accomplished by combining a mix of average and upper-bound exposures (USEPA 1989b, 1992b). The result of combining multiple upper-bound assumptions is that the final estimate of potential exposure and risk is health protective, and the majority of peoplewill have a lower level of potential risk.

The risk assessment approach used here employed a combination of upper bound and average assumptions consistent with guidance(USEPA 1989, 1990, 2014). Consequently, the resulting risk estimates are expected to be on the high end of the range of risks but within the range of plausible outcomes. For evaluation of potential exposure via consumption of LPRSA fish and crab, the following assumptions were used for the RME scenario:

- 90th percentile fish and crab consumption rate,
- 95 percent upper confidence limit on the arithmetic mean concentrations of chemicals in fish and crab tissue,
- No loss of chemicals from the fish or crab tissue due to cooking or consumption practices (i.e., the upper-bound assumption that fat and cooking juices are always consumed),
- All of the fish/crab consumed comes from the LPRSA,
- 90th percentile exposure duration,
- Mean body weight,
- Upper bound cancer slope factors.

The combination of these assumptions has produced estimates of potential risk that are likely to be well above the risk experienced by the average member of the potentially exposed populations. As stated in the Cancer Guidelines and other guidance documents, within a population a portion will be at the high end of the distribution while risks to the average individual represented by the 50^{th} percentile will be lower. This risk assessment found that the risks to the average individual (i.e., CTE scenarios) still remained above the risk range and/or the goal of protection of an HI = 1.